

THE MECHANISM OF INTESTINAL PERFORATION DUE TO DISTENTION*

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INTESTINAL perforation following prolonged over-distention is quite a different entity from perforation resulting from an intestinal ulcer. It is of much less common occurrence, and appears, so far as I have observed, only in cases of mechanical ileus. The perforation in such cases is generally situated upon the antimesenteric surface of the intestine, usually quite close to the point of obstruction. It is surrounded by an area of discoloration generally diamond-shaped or of an irregular shape resulting from the coalescence of two or more diamond-shaped areas. These diamond shapes correspond roughly to the terminal anastomotic tree of the intestinal vessels and are evidently the result of hemorrhage from these vessels; that is, hemorrhagic infarct of the area supplied. The mechanism of this infarction appears to be as follows: Distention of the intestine increases its diameter. Any increase in its diameter is tripled in its circumferential measurement. In other words, if the diameter of an intestine is increased by distention from 1 cm. to 3 cm., its circumference is at the same time increased from 3 cm. to 9 cm. Thus, the difference in the diameter is only 2 cm., while the difference in the circumference is 6 cm. A moderate increase in diameter, therefore, results in considerable stretching of the wall. The intestinal vessels pass between the layers of the wall along its circumference from their origin at the mesenteric border to their terminal anastomoses at the antimesenteric surface, becoming progressively more thin-

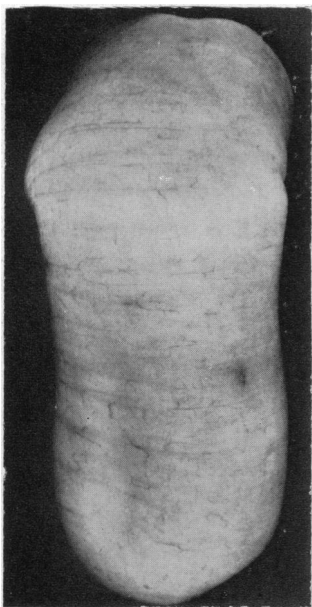


FIG. 1.—S. P. No. 7489. Intestine obstructed 144 hours. Small hemorrhagic areas in region of terminal intestinal vessels.

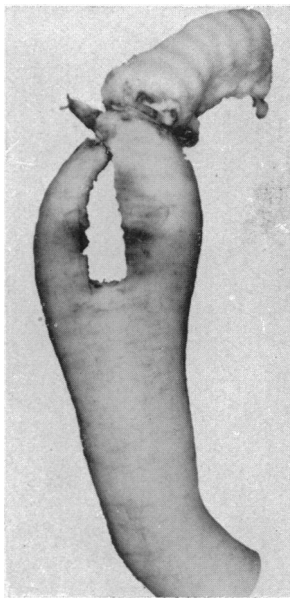


FIG. 2.—S. P. No. 5882. Intestine obstructed 72 hours. Diamond-shaped infarct on antimesenteric surface with section out for microscopic examination.

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walled and more narrow of lumen. Because they are elastic the stretching of the intestinal wall from distention still further thins the vessel walls and narrows the vessel's lumen like a stretched rubber tube. At the same time the pressure from within the intestine tends to flatten out the vessel's lumen.

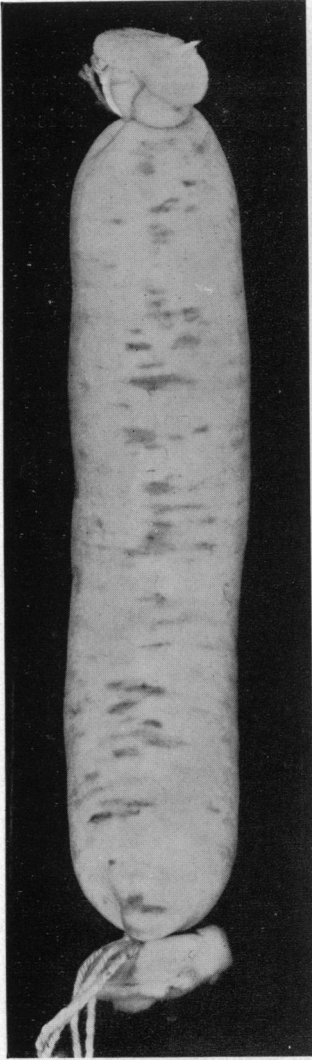


FIG. 3.—S. P. No. 7471. Intestine obstructed 77 hours. Multiple infarcts on antimesenteric surface.



FIG. 4.—S. P. No. 7471. Mesenteric surface of same specimen as Fig. 3. Absence of infarcts near mesentery.

The narrowing of the vessel's lumen and the thinning of the vascular wall are maximum at the antimesenteric surface of the intestine where the terminal anastomoses occur; and, the distention pressure being constant throughout the lumen of the intestine, the maximum effect is seen at the antimesenteric surface where a union of the three factors of pressure, thin wall and narrow lumen finally results in obliteration of the vessel. This obliteration occurs sooner in the vein than in the artery on account of the less resistant wall. The blood continues to pour through the arterial vessel until the pressure against the obliterated vein suffices to rupture the vessel

wall and permit extravasation and coagulation. Finally, the pressure occludes the artery as well as the vein. The area of tissue supplied by these terminal vessels is thus deprived of circulating blood and necrosis occurs. This necrosis is usually first evident in the submucosa and inner muscular coat, but rapidly extends to the other coats of the intestinal wall, and perforation

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may occur within twenty-four hours after the discoloration due to the hemorrhagic infarct has been first noticed.

It will be noted that hemorrhagic infarcts and perforations resulting from their necrosis cannot be expected to occur at any regular interval after obstruction, because the degree of distention varies so much in the individual cases and the infarction appears to depend entirely upon distention sufficiently prolonged and in excess of the normal limit.

The first twelve illustrations are of material from dogs operated upon in the laboratories of the Department of Surgery, College of Physicians and Surgeons, Columbia University. The last four are from a clinical case operated upon at the Presbyterian Hospital.

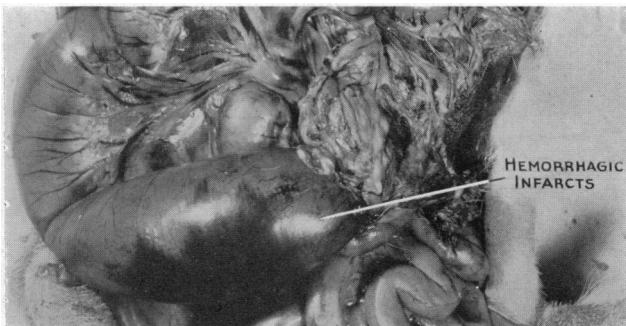


FIG. 5.—S. P. No. 5946. Intestine obstructed 180 hours. Coalescence of necrotic hemorrhagic infarcts into gangrenous area on antimesenteric aspect just above point of obstruction.

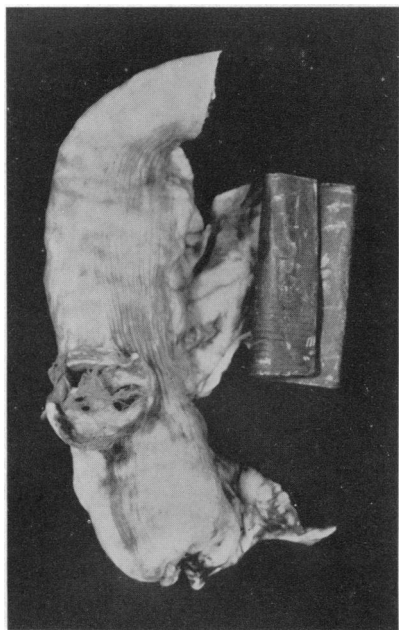


FIG. 6.—S. P. No. 7483. Intestine obstructed 120 hours. Perforation due to necrosis of hemorrhagic infarcts on antimesenteric surface.

Figure 1 shows an intestine that had been obstructed 144 hours. Several small hemorrhagic areas can be seen in the region of the terminal intestinal vessels.

Figure 2 shows quite distinctly the usual diamond-shaped form of the hemorrhagic infarct from which, in this instance, a section has been removed for microscopic examination. This intestine was obstructed for 72 hours.

Figure 3 shows multiple hemorrhagic infarcts in an intestine that had been obstructed only 77 hours. This specimen shows excellently their situation along the antimesenteric surface, while

Figure 4 shows the mesenteric border of the same specimen of intestine exhibiting no hemorrhagic areas near the mesenteric border.

Figure 5 shows a further development of multiple hemorrhagic infarcts three of which have coalesced to form an irregular discolored area on the antimesenteric surface near the blind end of the oral segment of the intestine. This specimen was taken from an intestine obstructed for about 180 hours.

Figure 6 shows a perforation near the blind end of the oral segment of an intestine that had been obstructed about 120 hours. The irregular diamond-shaped area of discoloration (which was very evident in the fresh specimen) around the perforation does not show distinctly in the photograph.

Figure 7 shows an interesting comparison of the pressure effects on thinning the intestinal wall, the two upper specimens having been obstructed only 48 hours, the two middle ones for 72 hours, the next to the lowest for about 96 hours, and the lowest for 180 hours.

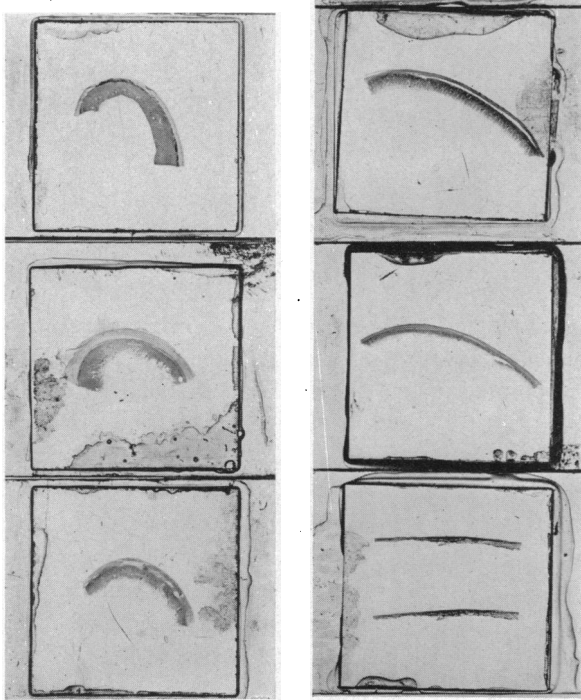


FIG. 7.—S. P. Nos. 5922, 5935, 5882, 5883, 5944, 5946. Intestines obstructed from 48 hours to 180 hours. Progressive stretching and thinning of intestine due to distention.

Figure 8 shows how the beginning of infarction probably appears microscopically where congested, thrombosed vessels are seen in the submucosa of an intestine obstructed for 72 hours.

Figure 9.—Extravasation has occurred from over-filled blood-vessels, both in the submucosa and in the muscular coat.

In Figure 10 the extravasation has increased in size, and the pressure is presumably greater.

In Figure 11 necrosis is well-advanced, and the integrity of the wall is gravely threatened.

In Figure 12 the completely necrotic area of the perforation is shown.

Figure 13.—Serosal surface of human intestine obstructed about 130 hours. Hemorrhagic infarcts may be seen at A, B and C.

Figure 14.—Same intestine photographed by transmitted light (somewhat similar to a radiograph). Hemorrhagic infarcts at A, B and C.

Figure 15.—Low power photomicrograph of section of same showing scattered submucous hemorrhages.

Figure 16.—Same showing single large submucous hemorrhage.

The last four figures are taken from the human case whose history follows: (I am indebted to Dr. F. B. St. John for the opportunity of reporting it.)

CASE I.—S. W., a woman of thirty-eight years, came to the Presbyterian Hospital on the night of October 2, 1923, with a history of abdominal pain which had begun seventy-two hours before and had shortly been followed by nausea and vomiting. The pain, though varying in intensity and moving from upper to lower abdomen, had persisted,

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and the vomiting had been repeated several times each day but had not assumed a fecal character. Her bowels had moved on day of onset but not since. A cathartic taken the day before admission had been promptly vomited, and an enema given on the day of admission had returned practically unchanged. She was worried by the fact that she had passed no flatus, but she complained of no undue abdominal distention. The only relevant items of her past history were an appendicectomy, four years previous, and an

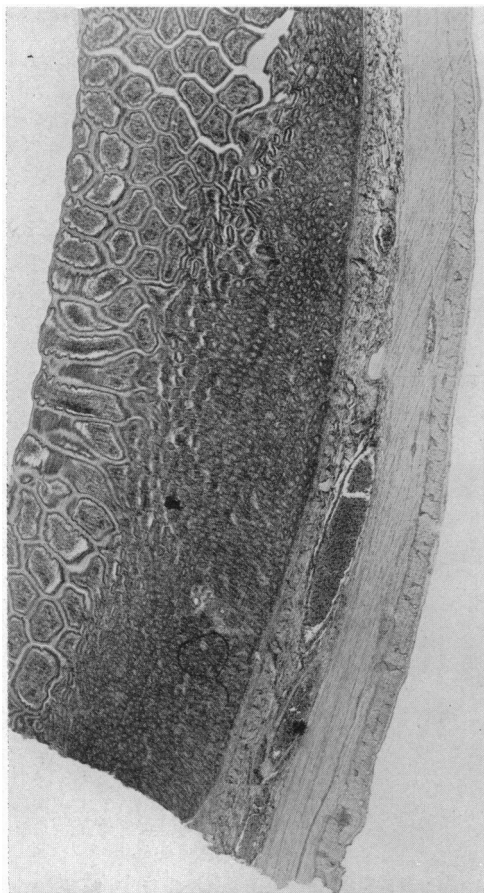


FIG. 8.—S. P. No. 5922. Intestine obstructed 48 hours. Thrombosed vessels in submucosa.

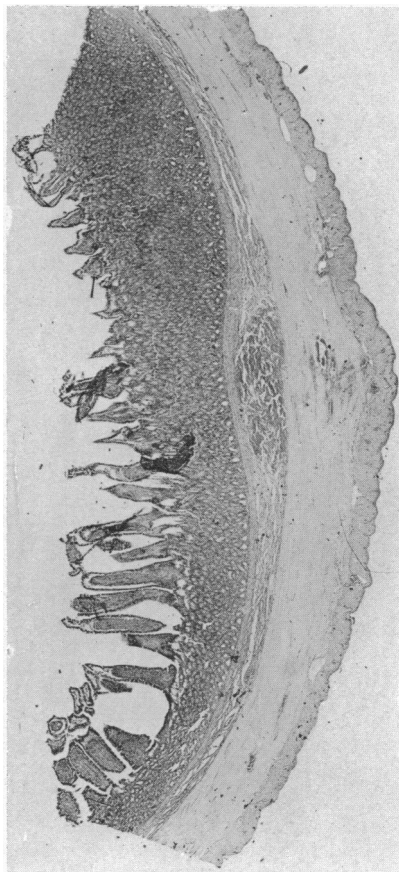


FIG. 9.—S. P. No. 7480. Intestine obstructed about 72 hours. Extravasation in submucosa and muscularis. Beginning of hemorrhagic infarct.

attack of abdominal cramps, nausea and vomiting that lasted twenty-four hours, one year previous to onset of her present illness.

On admission, she was apparently in some pain but did not appear to be very ill. Her pulse, blood pressure, temperature, and respiration, were within normal limits, and her urine was normal. Her white blood count was about 14,000, polymorphonuclears 80 per cent. Heart and lungs negative except for a few râles at left apex posteriorly. The abdomen was not distended and moved with respiration. Its lower half was rather more prominent than the upper and was less tympanitic on percussion. There was no visible peristalsis and no stiffening of the gut was felt. A small oblique scar in right lower quadrant indicated the old appendicectomy. There was slight tenderness, both direct and release, more marked to left of midline and slight muscle spasm which was thought to

be chiefly voluntary. There was slightly increased discomfort when uterus was moved in pelvic examination. There were no feces in rectum. Otherwise, the physical examination was negative. A diagnosis of partial intestinal obstruction due to adhesion was made, and as she appeared to be in good condition and had recovered spontaneously from her previous attack a year before, palliative treatment by poulitices, enemas, colon

irrigation, and hypodermoclysis was begun.

During the first 48 hours after admission, 5 out of 6 colon irrigations given, brought away feces and flatus. Her temperature, except for one observation, remained below 100° F., her pulse below 90, her respiration below 20. Her white blood count fell to 9,000, polymorphonuclears 61 per cent. She vomited several times, however, her abdomen became distended, her urine showed a trace of albumen; her blood urea was found to be quite high and her general condition looked less favorable. On the morning of the third day (almost 60 hours after admission and 130 hours after onset) Dr. Barclay Parsons operated under ether anesthesia. Upon entering the peritoneal cavity, greatly

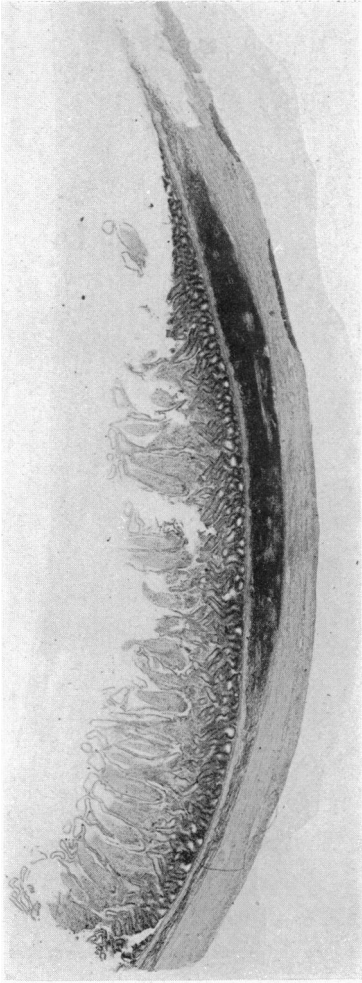


FIG. 10.—S. P. No. 7471. Microscopic section from specimen shown in Fig. 3. Large extravasation. Wall much thinned by pressure.

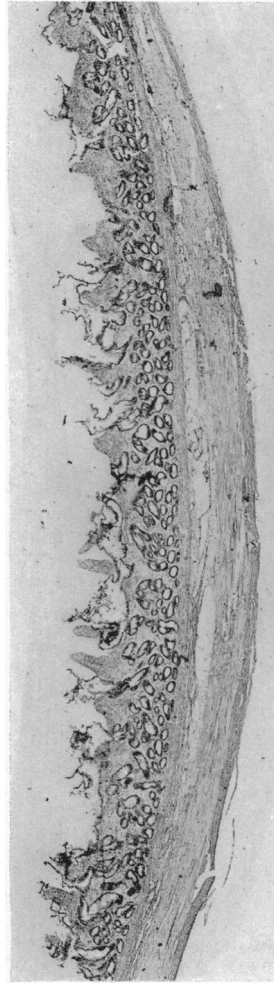


FIG. 11.—S. P. No. 7489. Microscopic section of specimen shown in Fig. 1. Advancing necrosis of infarcted areas.

distended injected loops of swollen intestines presented. There was no free fluid. In the right lower quadrant there was a broad band of adhesions extending upward from the cæcum and terminal ileum to the anterior abdominal wall just below the old scar. There was no evidence of kinking here, and no constriction of the intestines. Upon exploring the ileo-cæcal region, however, the intestines were found to be bound down as if by internal herniation behind in the ileo-cæcal fossa. Manipulation freed the loop, which presented a constriction proximal to which the gut was greatly distended, reddened, and of paper-thin consistency. Distal to the constriction, the gut was collapsed, but the great discrep-

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ancy in diameter diminished rapidly while the loop was being examined. While exploring the intestine a fecal odor was noticed, and fecal contents were discovered leaking from an opening in the distended gut. This opening was about 1 cm. in diameter, resembled a tear rather than an ulceration, and was situated about half way between mesentery border and antimesenteric surface. The mesentery in this region was very much injected



FIG. 12.—S. P. No. 7483. Microscopic section of specimen shown in Fig. 6. Completely necrotic edge of perforation.

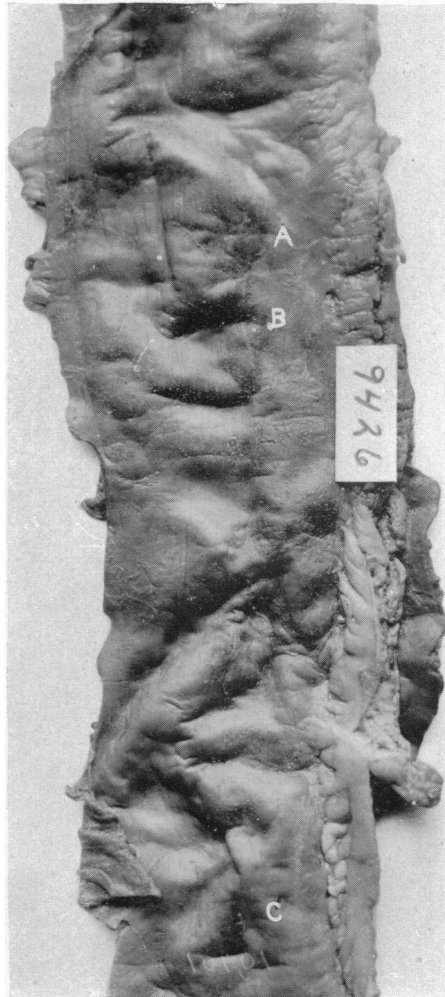


FIG. 13.—P. H. Autopsy No. 9426. Hemorrhagic infarcts at A, B and C on antimesenteric aspect of intestine which has been split open along its mesenteric attachment.

and somewhat thickened, but there were no noticeably enlarged lymph-nodes. A short distance away, the serosa of the gut split for a distance of 2 cm. merely when pressure was exerted upon an adjacent loop. There was no pathology noted other than the distended, congested, friable intestine above the point of obstruction, and the collapsed intestine distal to it.

The split serosa was repaired, the rim of the perforation in the intestine excised, and the aperture closed. An enterostomy was made and the wound closed with drainage.

Microscopic examination of the specimen excised showed that the mucosa was intact in many places. In other places the epithelium was absent, and both mucosa and submucosa showed hemorrhage. The submucosa and muscularis were cedematous and in places showed degeneration and hemorrhage, and all layers were infiltrated with

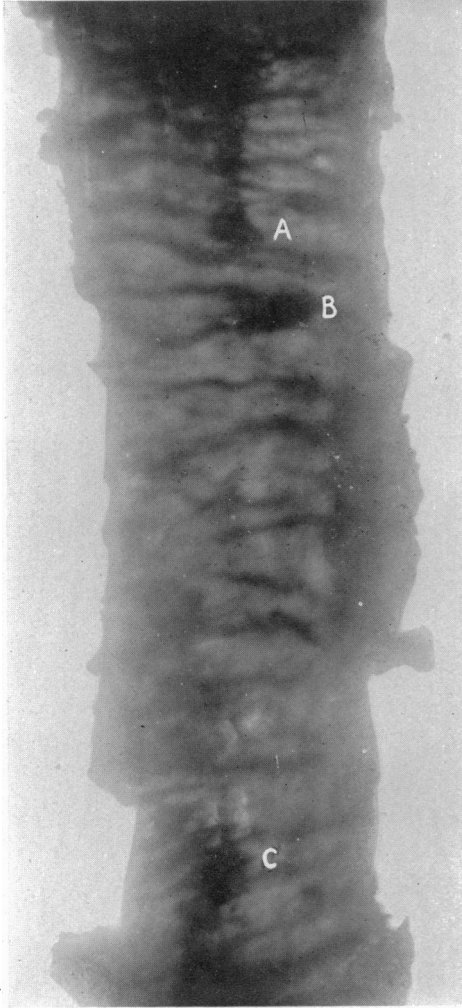


FIG. 14.—Same specimen as in Fig. 13, showing infarcts by transmitted light at A, B and C.

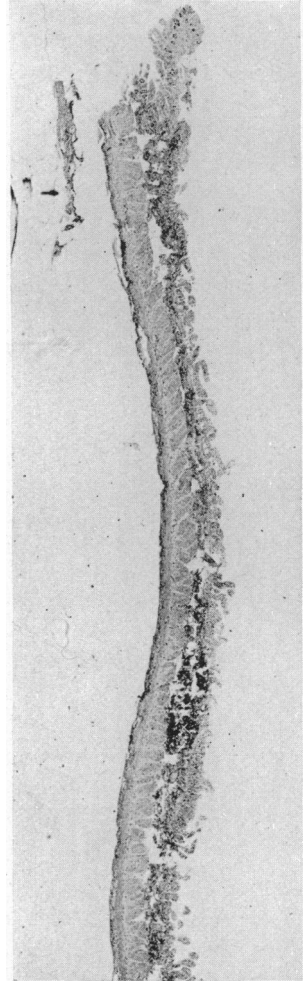


FIG. 15.—P. H. Autopsy No 9426. Low power of intestine showing very small hemorrhagic infarcts in submucosa.

leukocytes. The patient died about five hours after operation, with signs of acute respiratory and cardiac failure.

At autopsy, a pulmonary embolus was found and the intestine showed the striking changes due to over-distention resulting in hemorrhagic infarction. One of the areas of infarction had presumably necrosed to the point of perforation, but it was not definitely determined whether perforation had been spontaneous or had been induced by the necessary handling of the gut in freeing the obstructed portion.

The peritoneal cavity contained an excess of thin, bloody fluid most abundant in the pelvis. The omentum lay for the most part in the right half of the abdomen. Where

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the loops of small intestine are exposed they are lightly bound to the parietal peritoneum by a thin fibrinous exudate. The small intestine is moderately distended with gas; the surfaces are covered with thin fibrinous layer of exudate. The vessels in places are injected and there are occasional small fresh hemorrhages beneath the serosa. The transverse colon is somewhat distended. The stomach extends over to the costal margin at the right mid-axillary line and completely hides the lower border of the liver.

Gastro-intestinal Tract:—The stomach is negative except for post-mortem erosion. The duodenum is negative. There are no hemorrhages beneath the mucosa or serosa. It appears normal in every respect. The intestine: the serosa of the small gut is covered with fibrinous exudate, and in places the exudate is fibrino-purulent. Beneath the serosa, particularly in the first part of the jejunum, one sees dark areas which are apparently hemorrhagic and which, in general, follow around the circumference of the gut. At one point 135 cm. from the beginning of the jejunum, there is an enterostomy wound with the tube sutured into the gut. It was this tube that passed out through the omentum. Twenty-five cm. above this tube a line of sutures in the gut runs parallel to the mesenteric attachment, and here there appears to have been an opening extending directly into the lumen (probably the perforation noted at operation). There is no change made out around this area except hemorrhages beneath the margin of the mucosa. In the first part of the jejunum the mucosa appears normal except for occasional small hemorrhages lying beneath it. As one passes down the mucosa there is no exudate seen on its surface;

it has rather a velvety appearance. In a few places the crests of the rugæ appear injected, and this injection is particularly marked where the enterostomy tube was inserted. Here the submucosa is rather hemorrhagic, but the mucosa seems to be intact, and no exudate is made out. Extensive hemorrhage is seen beneath the mucosa about 25 cm. beyond the enterostomy opening, but there is no exudate seen on this part. In the ileum there is seen beneath the serosa a narrow, hemorrhagic band about $\frac{3}{4}$ mm. wide, which entirely encircles the gut. The serosa is roughened over this area, as if there had been an adhesion (this was point of obstruction). Within the gut, the mucosa directly

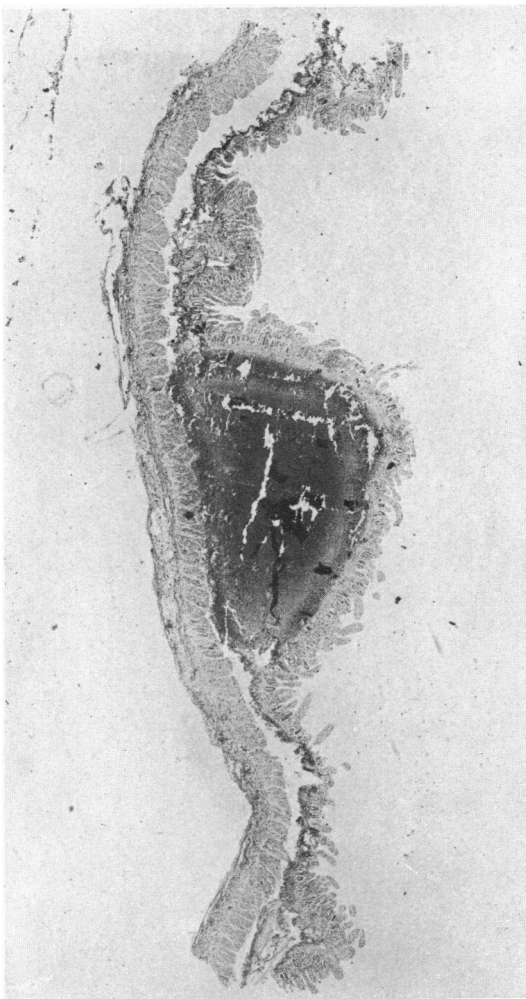


FIG. 16.—P. H. Autopsy No. 9426. Low power of intestine showing larger submucous hemorrhage.

overlying this band has disappeared and there is a grayish exudate encircling the gut which corresponds exactly to the outline of the hemorrhagic band seen beneath serosa. This ulceration is seen 100 cm. below the enterostomy opening, or 235 cm. from beginning of jejunum and 200 cm. from ileo-cæcal valve. The vessels in the mesentery at the site where this encircling ulceration of the intestine occurs do not seem to be thrombosed. It passes through a large Peyer's plaque. The gut below this area is quite dark red in color. The mucosa is quite purplish but seems intact. The Peyer's patches and solitary follicles stand out very sharply. Just at the margin of the ileo-cæcal valve is an area which appears to be slightly ulcerated, but this cannot be definitely determined. This area is only about 1 cm. in greatest diameter. The mesentery of the small intestine, especially that portion going to the ileum, is covered with fibrinous exudate. There are numerous recent hemorrhages beneath the peritoneum, and in the smaller veins are dark red thrombi.